TESTIMONY: Prof. Ellen K Silbergeld

EPA Hearings on Regulation of Utility Mercury Emissions
Philadelphia 25 February 2004

I am Ellen K. Silbergeld, Professor of Environmental Health Sciences and Epidemiology at the Bloomberg School of Public Health, Johns Hopkins University, in Baltimore Maryland. I am appearing without compensation as a private citizen, at the invitation of the Sierra Club, and my testimony is based upon my research experience on the toxicology and epidemiology of mercury compounds, as well as my experience in regulatory risk assessment and risk management, including the application of "cap and trade" mechanisms to achieve goals in reducing air pollution. My background and training are outlined in the attached documentation; my PhD is in environmental engineering sciences from Johns Hopkins School of Engineering, and I have held research positions with NIH and the University of Maryland Medical School. I have served as a member of EPA's Science Advisory Board as well as an advisor to the Department of Energy, the CDC, the World Health Organization, the World Bank, the Pan American Health Organization, the National Toxicology Program, the National Academy of Sciences, and many other international, national, and state commissions and expert committees. I was a member of EPA and NIH committees evaluating the sources and risks of mercury exposures and I participated by invitation in the deliberations of the NRC Committee on the Toxicology of Methyl Mercury. I am currently directing funded research in my laboratory on mercury compounds, studying exposures and mechanisms of both organomercury compounds (including methylmercury and thimerosal) and inorganic mercury. Last year we published two major research papers: an epidemiological study reporting that adults may be as sensitive as young children to the neurotoxic effects of methylmercury exposure, via fish consumption; and one of the first studies to show that very low doses of mercury can accelerate autoimmune disease, in an animal model of lupus.

In this testimony I want to make three points, relevant to important aspects of your deliberations: (1) mercury compounds must be considered toxic air pollutants; (2)

exposures to mercury compounds are a serious and significant health concern for millions of Americans; and (3) it is dangerously inappropriate to propose a "cap and trade" policy for controlling the major remaining anthropogenic sources of mercury in the US.

Mercury compounds are toxic air pollutants. Mercury compounds are widely recognized as one of the most serious public health risks world wide, particularly for children (see WHO 1990 report; NRC 2000 report). Mercury compounds can affect many organ systems, including the nervous system, kidney, heart, and immune systems. *However, we have not fully appreciated the range and severity of mercury toxicity.* Public health policy, including the risk assessments conducted by federal and state agencies, has appropriately focused on the developing nervous system as a very sensitive target for irreversible toxic damage. However, mercury has multiple effects of many organ systems in addition to the developing brain. We recently published an epidemiologic study indicating that adults exposed to methyl mercury via fish are also at risk for neurocognitive deficits, with a dose:response relationship very similar to that found for children exposed prenatally (Yokoo et al 2003):

Table 3: Regression coefficients β of adult's hair mercury concentration as a predictor of neurobehavioral test results.

Test	β*	95% CI	β**	95% CI
Fine Motor Speed	-3.40	-5.80, -1.00	-3.20	-5.40, -1.00
Digit Span	-0.14	-0.29, -0.001	-0.15	-0.29, 0.003
Digit Span backward	-0.09	-0.18, -0.001	-0.09	-0.19, -0.009
Digit Symbol	-1.21	-2.08, -0.33	-0.54	-1.2, 0.16
Easy Learning	-0.37	-0.70, -0.04	-0.34	-0.64, -0.04
Difficult Learning	-0.21	-0.42, -0.001	-0.15	-0.34, 0.03
Logical Memory first story	-0.29	-0.51, -0.09	-0.27	-0.49, -0.06
Errors of commission	1.39	0.26, 2.5	1.45	0.28, 2.6

* – not adjusted; ** – adjusted by age, gender and education level

In addition, recent research in our group and elsewhere has identified the cardiovascular system and the immune system as important targets for mercury toxicity across the lifespan. Because these studies have been published since the 2000 NRC report and risk assessments by FDA and EPA, I will review these data here. In follow up studies in Minimata and in the Faeroes study of children exposed perinatally to methyl mercury via fish consumption, alterations in cardiovascular function have been reported (Oka et al 2002; Sorensen et al 1999). In 2003, my colleague Dr Eliseo Guallar reported that

mercury exposures were associated with cardiovascular disease in adults. In this elegant analysis, Guallar et al (2002) demonstrated that consumption of fish containing mercury resulted in loss of the beneficial effects of fish consumption for cardiovascular function, that is, the methyl mercury ingested by fish consumers abrogated the recognized benefits

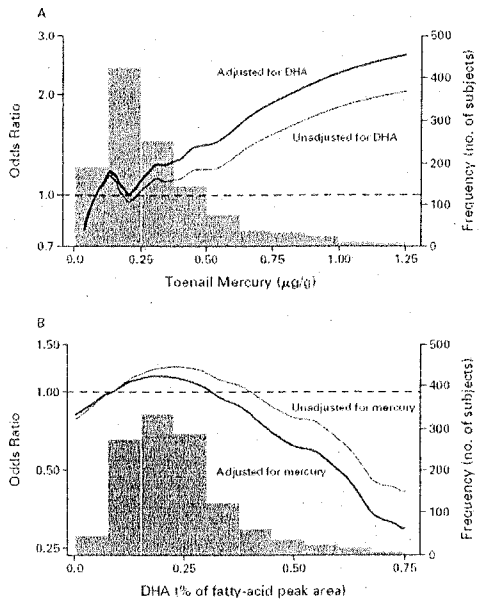


Figure 1. Nonparametric Estimates of the Risk of Myocardial Infarction According to the Levels of Mercury in the Toenails (Panel A) and of Docosahexaenoic Acid (DHA) in Adipose Tissue (Panel B).

All curves have been adjusted for age and center. The nonparametric regression models used a lowess smoother with 40 percent span.[9] The reference value (odds ratio = 1.0) was set at 0.08 μg per gram for mercury and 0.08 percent of the total fatty-acid peak area for DHA, both values corresponding to the 5th percentile of their respective distributions among controls. The bars represent the frequency distribution of mercury and DHA in the study sample.

of consuming omega-3 fatty acids of which fish are an excellent source.

The immunotoxic effects of mercury have long been reported in experimental studies, many conducted by researchers here in Philadelphia (Prof Shenker, Monestier, and Kono). These researchers and others have shown that administration of mercury compounds to rats and mice can induce autoimmune dysfunction similar to that observed in such autoimmune diseases as lupus and scleroderma. However, there has been little data to suggest that mercury could cause autoimmune disease in humans. We have examined these potential risks of mercury in a different way, to test whether mercury can accelerate autoimmune disease in the context of triggers of these diseases, such as genetic

susceptibility, infection, or exposure to antigens. We reported last year that pretreatment of mice with very low doses of mercury can accelerate and exacerbate lupus in an animal

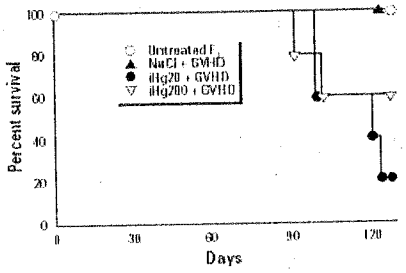


Figure 1. Pretreatment with iHg accelerates mortality in chronic GVHD mice ($n = 5$ for each treatment group). See "Materials and Methods" for details.

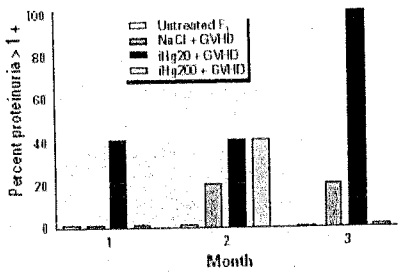


Figure 2. Pretreatment with iHg accelerates the appearance of proteinuria in chronic GVHD mice. Proteinuria was tested at the times indicated, as described in "Materials and Methods."

model of disease, resulting in premature mortality, more extensive kidney damage, and more rapid dysregulation of the immune system (Via et al 2003).

To put our experiments in perspective, we are exposing our mice to doses equivalent to consuming one can of tuna fish per day with a concentration of 5-10 ppm methyl mercury. In our current research we are examining interactions of low dose mercury with infections, such as Coxsackie B virus, which are major causes of autoimmune cardiomyopathy in humans. Again, we found that mercury accelerates and worsens heart disease in the context of viral "priming"(Nyland et al 2004). Autoimmune myocarditis is a leading cause of sudden heart failure in young persons; the possibility that mercury exposures could uncover latent disease, or worsen disease, is very serious.

Based on these studies, and the continued research on mercury worldwide, it is fair to say that we have not yet fully comprehended the range of mercury toxicity and its risks for human health. In many ways, we are still at the point in evaluating mercury as a toxic air pollutant as we were in thinking about lead some 25 years ago. We know that mercury is

dangerous, and we know some people may be excessively exposed. However, we do not fully appreciate its toxicity and hence we cannot disregard the range of exposures current in the US population.

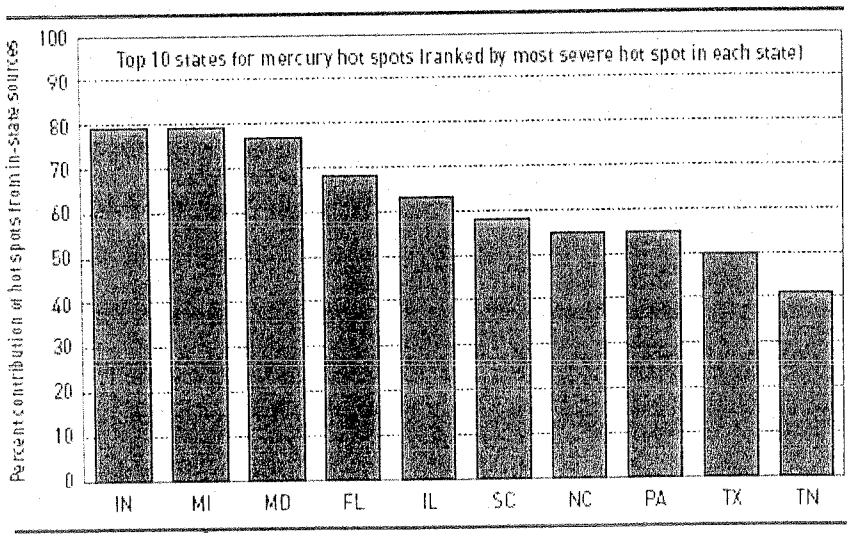
Exposures to mercury compounds are a significant threat to millions of Americans. One yardstick by which to judge the need for urgent interventions in a public health problem is to evaluate current levels of exposure to a toxic agent like mercury. Several recent analyses have been undertaken on exposures of the US population to mercury compounds, most recently by Dr Kathryn Mahaffey and her colleagues at EPA. (Their report is available on line from Environmental Health Perspectives, the scientific journal published by NIEHS). Mercury exposures can be evaluated either by population studies of mercury concentrations in blood or hair, which was done by the CDC in 2003 (Schober et al 2003). Exposures can also be determined by analyzing mercury concentrations in food, which is the major source of exposure for the US population. Mahaffey and colleagues have updated the earlier assessment of US exposures, using information on blood mercury levels and on diet. Their analyses support the urgency of taking comprehensive and effective actions to reduce ongoing inputs of mercury into the environment. For all US women of childbearing age, half have blood mercury levels in excess of 0.94 micrograms/L. Nearly 10% have blood mercury levels greater than 5 micrograms/L, with a range of 2.7 to 25% depending upon ethnicity. The NRC recommendations in 2000 supported a reference dose for mercury in cord blood of 5.8 micrograms/L. *Mahaffey et al estimate that more than 300,000 infants may be born each year to women whose blood mercury levels are in excess of this health based guidance.* Clearly, this is an environmental health issue demanding rapid intervention.

Mercury comes from many sources, natural and anthropogenic, and each individual is exposed to the sum of all these sources. For most Americans, the proximate source of mercury exposure is through the food supply, primarily through seafood. Finally, the FDA seems ready to adopt the current risk assessment, developed by the National Research Council and adopted by EPA. However, this is the proximate source of mercury, and attempting to reduce exposure by controlling the foods we eat is an inefficient and ultimately uncertain public health policy. Moreover, without controlling the ultimate sources of mercury, we are essentially writing off seafood as a food source.

The ultimate source of mercury is overwhelmingly from energy production using fossil fuels. Prudent and effective public health policy requires that we examine options for controlling this source, rather than eliminating seafood and some freshwater fish from our diets for now and forever.

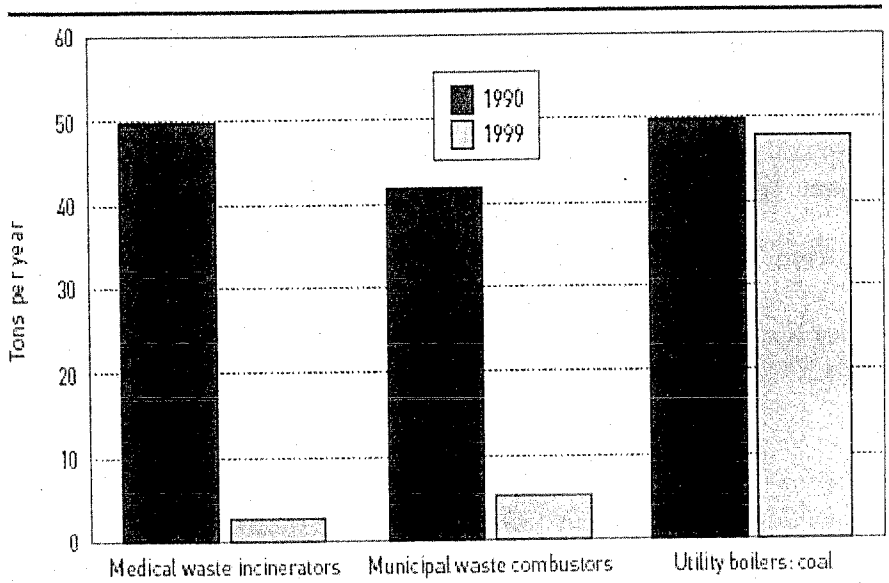
"Cap and trade" policies are not appropriate for mercury. I am proud that I worked for the environmental organization Environmental Defense that has developed innovative strategies for protecting our environment and human health. One of these strategies has been the careful selection and implementation of so-called "cap and trade" policies for certain pollutants, notably sulfur oxides. From this experience, there are criteria we can apply in determining what policies are appropriate for controlling specific pollutants. First, trading only works to prevent environmental impacts and harness efficient private sector mechanisms under the following conditions: (1) it doesn't matter where the pollutant is released, so that if one source accumulated "trading rights" and emits more pollution than a source that sells these rights, there will be no local impacts around the buyer source. (2) the pollutant should not accumulate in the environment, such that continuing emissions do not build up in ecosystems or food pathways. (3) the current levels of exposure should be acceptable such that it is not necessary to implement a rapid overall reduction in exposures at the local or national level.

None of these conditions are met in the case of mercury. *It does matter where mercury is emitted.* In an analysis of EPA data conducted by Environmental Defense, it was shown that in many states with mercury problems (evidenced by fish advisories) local sources



Source: Draft Mercury Deposition Modeling Results, EPA Office of Water, 2003.

are the cause of environmental "hot spots". It these sources utilize trading rights, then the problem of local "hot spots" will continue. This is likely, since the reason for these hot spots is current levels of release, reflecting the fact that it is more convenient, economically and technologically, for these sources to emit mercury rather than control their facilities. *Mercury accumulates in the environment and in food pathways affecting wildlife and humans.* Mercury is an element and thus never disappears. In addition, in the aquatic environment, inorganic mercury emissions are transformed by bacteria into methyl mercury, which is bioaccumulated by organisms through complex food webs resulting in concentrations of methylmercury in large fish that eat other fish tens of thousands of times higher than the concentrations in water or sediments. *Current levels of exposure are unacceptable.* For that reason, it is imperative for us to take action to reduce mercury exposures from all sources, but most expeditiously to reduce the largest and least controlled sources. We have the technology to control utility emissions, as has been demonstrated in this country for other combustion sources and in Europe for utility plants. Data below show the dramatic reductions achieved by waste incinerators.



We do not have room for trading, when hundreds of thousands of adults and babies are at risk because of current levels of exposure. We do not have time for trading, when consumers must choose between a healthy diet, incorporating seafood, and avoiding the hazards of mercury for themselves and their children.

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